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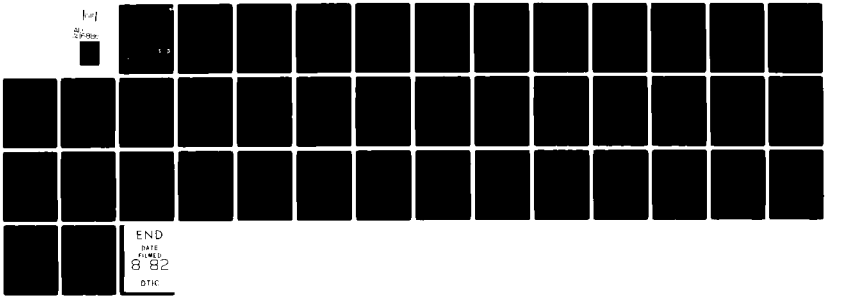

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NEURON SELECTIVITY: SINGLE NEURON AND NEURON NETWORKS*

L. N Cooper, P. Munro, and C. Scofield

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ABSTRACT

A theory for neuron selectivity in visual cortex is presented and compared with neurophysiological data. In this theory, a neuron becomes maximally selective over the set of patterns presented to it. Theoretical predictions correspond well with results of various experimental paradigms including normal rearing, monocular and binocular deprivation, and reverse suture. Extensions of the theory to cortical networks based on anatomical data are discussed. The ideal synapse, which can alter its function between excitatory and inhibitory modes, must be resolved into excitatory and inhibitory synapses to agree with anatomical observation. A mathematical appendix provides stability analysis of the model's fixed point behavior.

INTRODUCTION

Several models for postnatal development in area 17 of visual cortex have been put forward previously (e.g. von der Malsberg, 1973 ; Perez et al., 1975 ; Cooper et al., 1979). The theory presented here describes not only the selective tuning of neurons in this area to specific visual stimuli, but also the interaction of visual signals from both eyes. Analytical studies and computer simulation indicate that response characteristics of model neurons evolve as a function of the visual environment in a manner consistent with experimental studies from several laboratories.

In the formulation of theories describing neural processing of any kind, certain common elements must be considered, for example the connectivity of the neuronal network. Consider a network (fig. 1) in which one population of neurons d projects to another population c. The net connectivity between the j-th cell of the input population (d_j) and the i-th output cell (c_i) is given by the value M_{ij}. A typical connectivity, M_{ij}, represents the net influence of several synapses, both in parallel and in series, both excitatory and inhibitory, possibly involving interneurons. M_{ij} represents the efficacy of an 'ideal synapse' (Nass and Cooper, 1975), a useful concept to the mathematician, but one which falls short of ideal in the anatomist's eyes. The idea is that each input axon firing frequency is converted to some net postsynaptic membrane depolarization in a given cell in the target population (c), and eventually will influence the firing of the cell.

[Figure 1 about here]

Mathematically speaking, the synaptic array M operates on the stimulus vector d producing the response vector c (Eqn 1).

$$c = Md \qquad [1]$$

Similar assumptions have been described in the two previous talks (Anderson ; Kohonen). In effect they have said that the connectivity matrix M can evolve to function as a memory. By 'memory' we generally mean that the synaptic strengths M_{ij} contain information in that they yield 'appropriate responses' in the postsynaptic population c to certain patterns of afferent activity. Rigorous mathematical theorems can be proven to show that such memory systems work. Kohonen et al (1977) used facial photographs encoded into a string of darkness (brightness) values each representing a small element of the visual field. The coded information from each pattern is distributed over all synaptic junctions in the network. This example is a beautifully graphic demonstration of what is meant by a distributed memory. This idea has the important quality that if any small portion of these junctions is lost, no specific faces are lost - rather the signal-to-noise ratio is reduced.

The principles which determine the evolution of a synaptic network with experience have to be formulated carefully to yield a system which is capable of both extracting information from the environment and accessing that same information at a later time. Ideally, the postsynaptic population c will

learn to respond appropriately to inputs d depending upon whether or not similar patterns have already been presented and, if so, in what context. Various types of modification rules yield memories which are suited to a variety of tasks. Over the last several years our group has developed a model of binocular orientation-selective cells in visual cortex by refining a set of equations for synaptic plasticity (Nass and Cooper, 1975; Cooper et al, 1979; Bienenstock et al, 1982) and is based on a diverse collection of physiological data.

REVIEW OF RELEVANT EXPERIMENTAL DATA

Since the original work of Hubel and Wiesel (Hubel and Wiesel, 1959) that led to the realization that visual cortical cells respond in a highly specific fashion to visual stimuli. These studies have been extended to the development of this specificity in the newborn animal. Michel Imbert and his collaborators (Imbert and Buisseret, 1975) have classified visually responsive cells into three groups - aspecific, immature and specific - according to their response properties and receptive field arrangements (figure 2). [Figure 2 about here] Aspecific cells are characterized by rather indiscriminant responses to circular stimuli moving in any direction across their receptive fields. The receptive field is usually rather large in size and circular in shape. Immature cells are more highly tuned in their response: they characteristically respond best to a correctly oriented rectilinear stimulus moving across the receptive field, and there is always an orientation for the stimulus that will evoke a negligible response. Their receptive fields are large, but more rectangular than the receptive fields of the aspecific cells. The most discriminant of all cells, the specific cells, show a sharp response

to an optimal orientation of the stimulus and are much less responsive to other orientations. These cells have receptive fields that are smaller in size than those of the two previous cell classes, and are rectangular in shape.

The evolution of these different cell types has been studied for kittens raised in different visual environments (Fregnac and Imbert, 1977, 1978; Fregnac, 1978). The results for kittens raised in a normal visual environment (NR) and for those raised in complete darkness (DR) since the first or second day of age are compared in figures 3 and 4. This data indicates that in the earliest stages of development there are some cells present that exhibit specific cell properties. However, visual experience is critical in the development of these cells. The normally reared animals show a pronounced increase in the number of specific cells relative to aspecific cells, while dark reared animals show just the opposite. The high degree of malleability of cortical cells in this critical period from about day 17 to day 70 is illustrated in figure 5. We see that as little as six hours of visual experience at 42 days of age can dramatically alter the ratio of specific to aspecific and immature cells (Buisseret et al., 1978).

[Figures 3, 4a, 4b about here]

[Figure 5 about here]

In addition, ocular dominance appears to depend on visual experience. It is found that before the age of 21 days, independent of visual experience, most immature and specific cells are driven optimally by the eye contralateral to the hemisphere in which they are located. After three weeks of age, normally reared animals exhibit a significant increase in binocularly driven cells (Fregnac and Imbert, 1978). This result is also found for animals that

are dark reared (Blakemore and Mitchell, 1973; Imbert and Buisseret, 1975; Blakemore and Van Sluyters, 1975; Buisseret and Imbert, 1976; Leventhal and Hirsch, 1980; Fregnac and Imbert, 1978). However, in animals whose eyelids have been sutured at birth (and thus deprived of binocular pattern vision, BD), a higher proportion of selective cells are found compared to dark reared animals and the proportion of binocular cells is less than normal (Wiesel and Hubel, 1965; Blakemore and Van Sluyters, 1975; Kratz and Spear, 1976; Leventhal and Hirsch, 1977; Watkins et al., 1978).

Finally, one of the clearest examples of the dependence of cortical development on the environment occurs when an animal is raised with one eye in competitive advantage over the other. When monocular lid suture (MD) is performed during the critical period, a rapid loss of binocularity to the profit of the open eye occurs (Wiesel and Hubel, 1963, 1965). If the sutures are reversed (RS), the closed eye opened and the experienced eye closed, it is possible to observe a complete reversal of ocular dominance (Blakemore and Van Sluyters, 1974).

FORMULATION OF THE THEORY

The theory we now describe deals with the development of selectivity and ocular dominance. The pattern environment available to the neuron consists of impulses directly associated with a specific receptive field on the retina and secondary lateral influences from cortical cells having different primary receptive fields which may overlap to varying degrees. One can easily imagine how synaptic delays can account for the translation of a spatio-temporal pattern such as a moving bar to a near-simultaneous, purely spatial pattern of postsynaptic potentials at a neuron's dendritic surface. Hence, the notion of a visual stimulus should be considered in its most general sense.

Refraining from choosing completely ad hoc assumptions, we first consider what aspects of a given synapse's environment might influence its development. Changes in the physical structure of either the presynaptic or postsynaptic elements or the cleft itself, as well as the chemistry of the synapse's immediate neighborhood come to mind as primary candidates. Of course consideration of proposed biochemical mechanisms (e.g. Changeaux et al, 1973) can be most useful at this stage of model development.

Let us begin developing our model with a formal statement of the functional dependence of dm/dt (Eqn 2). Namely, we assume here that the change in efficacy of a given synapse depends on the efficacy itself, both

the presynaptic and postsynaptic activities, as well as a time average of the postsynaptic actvity. Global factors may well play a strong modulatory role (see for example Kasamatsu and Pettigrew), but we do not consider them here.

$$\dot{m} = \dot{m}(m,d,c,\bar{c}) \qquad [2]$$

Two linear assumptions are made in this model. First, the transfer function (Eqn 3a), or input-output relation, is simply an inner product. More complicated functions have been tested and do not seem to have a qualitative influence on the results as long as they are monotonically increasing in both m and d. The modification function (Eqn 3b) includes the second linear assumption, namely that dm/dt is linear in d. Neglecting the second term, we see that the model relies on the premise that the change in the synaptic vector is parallel to the input, hence the response characteristics of the cell are influenced for that input more than any other possible stimulus of the same magnitude.

$$c = \sum_i m_i d_i \qquad [3a]$$

$$\dot{m}_i = \phi(c,\bar{c})d_i \qquad [3b]$$

This model is predicated on the notion that it $\phi > 0$ for a given input and

the evoked response then the m_{ij} change such that the cell's response to a future presentation of the same stimulus is enhanced. This idea was first put forward by Hebb (1949) so we will term such modification hebbian. Similarly a stimulus-response combination yielding a negative value for ϕ decreases the cell's affinity to that stimulus (anti-hebbian). The conditions on ϕ are illustrated in figure 6.

[Figure 6 about here]

Since we are modelling cells which become highly selective, we design a system which weakens responses to weak patterns and strengthens responses to strong ones. The strength of a neuron's response to a pattern is evaluated relative to a time-varying threshold value (θ) intrinsic to the cell, at which ϕ vanishes. This 'floating' threshold is linked to the expectation value of the cell's response evaluated with respect to the stimulus environment. If the time scale for synaptic modification is sufficiently larger than the usual duration of an input presentation, then this expectation value is close to a time-average.

If the threshold could not vary in time, the system would require some very specific assumptions (Cooper et al, 1979) in order to account for the highly tuned cells observed in visual cortex. By allowing θ to vary with the average response in a faster-than-linear fashion, the response characteristics evolve to maximum selectivity over the input environment. This spatio-temporal modification scheme forces θ and the nontrivial response values towards a common value such that the threshold is driven between them. When this occurs the response values below θ decrease toward zero and θ 'overtakes' the other values which have begun to

increase. Eventually only a small subset of the stimuli drive the cell above the modification threshold. In certain cases (see appendix), it has been proven that maximum selectivity is attained. Barring situations where some inputs have negative inner products (see Bienenstock, 1980), computer simulations indicate that the neuron is always driven to a maximally selective state.

The simple case of a neuron driven by two equiprobable two-dimensional stimuli provides a good example for examining these properties of the model. The input environment consists of two linearly independent vectors, d^1 and d^2, which are normalized and have a nonnegative inner product. They can be represented in a common vector space with the synaptic state vector m, since each afferent (a component in each d^i) corresponds to a unique ideal synapse (a component in m). We begin by seeking points which are candidates for stationary final states (fixed points), then we will look at the behavior of the system relative to these points. If we assume that the synaptic weights change slowly relative to the frequency of stimulus presentation, then we can assume that a fixed point represents any state for which the net modification induced by the two patterns is zero. Since the patterns are independent, dm/dt must be zero for <u>each</u> stimulus.

Let $\theta = c^2$ give the threshold value for the ϕ function. We see then that the locus of points for which $\phi=0$ for a given input (fig 7) is the union of two loci, namely a line corresponding to c=0 (Eqn 4a) and a parabola corresponding to $c=\theta$ (Eqn 4b). The intersections of these isoclines give the four fixed points, at which both patterns must give $\phi=0$. We define the order

[Figure 7 about here]

of a fixed point, s, to be the number (0, 1, or 2, in this example) of patterns which yield the response c = θ. Figure 7 divides the space into four regions, characterized by the sign of ϕ attributed to each pattern. Thus we see that the asymptotically stable final states are <u>fixed points of maximum selectivity</u>. An example of local stability analysis is given in the appendix.

$$m \cdot d^i = 0 \qquad i = 1,2 \qquad [4a]$$

$$m \cdot d^i = (m \cdot \bar{d})^2 \qquad i = 1,2 \qquad [4b]$$

DEVELOPMENT OF NEURONS UNDER VARIOUS REARING CONDITIONS: COMPARISON OF THEORY AND EXPERIMENT

The simulated behavior of neurons in visual cortex with binocular connectivity is illustrated in figure 8. The seemingly inconsistent experimental results (MD vs BD) are faithfully reproduced by computer simulation according to the model outlined here. Thus this theory gives one explanation which resolves the paradox. Each of these paradigms was tested in both deterministic and stochastic simulation algorithms over several pattern sets. The model withstood considerable noisy input, indeed successful simulation of some paradigms (RS in particular) required that a noiselike component accompany the 'pure' inputs.

[Figure 8 about here]

Binocular interactions do not play a special role in understanding the behavior of this theory with respect to either binocular deprivation or (correlated) normal rearing. That is, due to idealizing assumptions (for example, disparity between left and right inputs is not considered) these two cases drive the model neuron just as it would be driven if its connections were strictly monocular. Binocular stimuli presented in NR simulations were exactly correlated so that each pattern incident to the left-eye synapses was consistently accompanied by a corresponding pattern to the right-eye synapses. Thus each binocular input enjoys a one to one correspondence with a particular input to either eye. The left and right components of each pair used here are identical, as a consequence the cell tunes to the same pattern in each eye (Fig 8a). Binocularly deprived input environments consisted of stimulus

components uniformly distributed over some range with zero mean. In this case (BD), the average response of the cell is null and so o is always non-negative resulting in the synaptic state modifying randomly (Fig 8b).

The development of a neuron receiving patterned input from only one eye (and uniform noise from the other) is interesting. The naive prediction incorrect. Even though the response curve seeks maximum selectivity with respect to the open eye, the response to the other eye does not fluctuate randomly. Rather, as in the laboratory, the neuron becomes nonresponsive to inputs to the deprived eye (Fig 8c). Asymptotic convergence to this state is assured regardless of the initial state. The theoretical implications for the RS paradigm are as follows: A monocularly deprived neuron, having reached a monocular selective state is driven to another monocular selective state preferring the newly opened eye upon reversal of suture (Fig 8d).

These monocular results rely upon some activity, albeit purely random, to be present in the afferents from the closed eye. Such noise may be due to diffuse light through the eyelid or spontaneous firing of LGN and/or retinal neurons. As a neuron becomes selective with respect to the open eye, those few patterns which are preferred give a response near threshold whereas the other patterns give a much lower response. In either case ϕ is near zero. Noise accompanying a preferred pattern drives the neuron from the modification threshold, so the deprived synapses grow stronger. However, the opposite effect weakens the synapses when 'uninteresting' patterns are presented. A mathematical demonstration of this argument is given in the appendix.

THE MANY-NEURON PROBLEM

So far we have been discussing selectivity on the single unit level. However, cortical anatomy suggests that the geniculocortical component of the afferents to a cell is a small fraction of those afferents (Hornung and Garey, 1981). Thus it is very likely that cortico-cortical inputs play an important role in the development of neuron selectivity (Creutzfeldt et al., 1974; Sillito, 1975). The development of selectivity then might be viewed as a many-neuron problem.

Formulation of the many-neuron problem raises several issues. These may be classified into three broad categories. The first concerns relevant cortical anatomy. Neuroanatomical studies have provided extensive detail. We would like to include details relevant to the development of selectivity, yet keep the model to a manageable complexity. Second, the problem of response dynamics needs to be examined. The prescence of cortico-cortical loops could lead to 'resonance' effects in the response of a cell to a pattern. Thus the transfer function of the cell population must be more carefully defined than the single cell case (see equation 3a). Two possible solutions are (a) Cortical activity rapidly settles before the afferent message changes and (b) Cortical response to a pattern decays rapidly (Bienenstock et al., 1982). The

final class of problems concerns the evolution of synaptic strengths. Physiological studies have yet to clarity which cell types develop stimulus selectivity. Thus we need to make assumptions about the evolution of geniculocortical synapses on inhibitory and excititory cells. In addition, the role of plasticity in intracortical synapses is not clear.

The many-neuron problem has been treated before by other researchers. Von der Malsburg (1973) chose an anatomy of two cell types corresponding to excititory and innibitory cells. In his model, the two cell types were distributed equally but only the excititory cells had geniculocortical afferents. The problem of response dynamics was solved according to alternative (a) above. Finally, von der Malsburg chose an intracortical circuitry in which a cell received static synapses that were short range excititory and long range inhibitory.

Since this work, further experimental studies have emphasized the role of cortical inhibition in neuron selectivity (Sillito, 1975). However, evidence suggests that both inhibitory and excititory cells receive geniculate afferents (Davis and Sterling, 1979). Our model uses a circuitry incorporating excititory and inhibitory cells which both receive geniculate input. We assume alternative (b) above: Only monosynaptically and disynaptically mediated components of the geniculate message are integrated to give a cell's response. One of the results of incorporating a 'first order anatomy' into the many-neuron problem has been a resolution of the 'ideal synapse' into seperate excititory and inhibitory synapses. It can be shown that under proper conditions on the intracortical inhibitory synapses, and

assuming a non-selective inhibitory cell, excititory cells that evolve according to equation 3b will reach a state maximally selective with respect to their own individual environments. Thus, with respect to cortico-cortical connectivity, selectivity appears to depend more on inhibition than than it does on excitation. Further work on the precise spatial relationships and evolution equations of the intracortical circuitry is necessary before the mechanisms specifying global cortical properties will be fully understood.

APPENDIX

The function $\phi(c,\bar{c})$ that describes the post-synaptic effect on the evolution of the synaptic efficacies has zeros at c = 0 and at $c = \bar{c}^p$. For those values of c, the synaptic strengths will not change. The values of m for which $\phi = 0$ when $d \neq 0$, are called fixed points. The fixed points of m-space can be classified according to the following criteria. A fixed point of index s is a point in m-space for which $m^* \cdot d^j = (m^* \cdot \bar{d})^p \equiv \mu^{(s)}$, with $d^j \in \mathcal{D}^\mu \equiv \{d^1, \ldots, d^s\}$ and $m^* \cdot d^i = 0$ for $d^i \in \mathcal{D}^\circ = \{d^{s+1}, \ldots, d^K\}$.

The stability of a fixed point will be investigated, rather informally, by examining the time dependance of m if it is perturbed from the fixed point, m*. A point is unstable if after a small perturbation $m = m^* + x$, ($|x| << |m^*|$), m evolves away from m*. A point is stable if m returns to m* after the perturbation.

Under proper restrictions on ϕ, we can assume that ϕ is a linear function of its argument for m near a fixed point. Thus for c near zero, $\phi(c,\bar{c}) \sim -\alpha_\circ c$, and for c near $\bar{c}^p$, $\phi(c,\bar{c}) \sim \alpha_\mu (c - \bar{c}^p)$. In the special case of $\bar{c}$ equal to zero, we assume that $\phi(c,0) \sim \alpha c^2$. Where we define $\alpha_\circ = -\frac{\partial \phi}{\partial c}\Big|_{c=0}$, $\alpha_\mu = \frac{\partial \phi}{\partial c}\Big|_{c,\bar{c}^p}$ and $\alpha = \frac{\partial^2 \phi}{\partial c^2}\Big|_{\substack{c=0 \\ \bar{c}=0}}$. These assumptions greatly simplify the analysis of the local stability of a fixed point.

The environment of the cell is assumed to consist of K linearly independent vectors $\mathcal{D} = \{d^1, \ldots, d^K\}$. Thus we define the average vector:

$$d = 1/K \sum_{\ell=1}^{K} d^\ell .$$

Then for $d^i \in \mathcal{D}^\alpha$, $d^j \in \mathcal{D}^\mu$:

$$m^* \cdot d^i = 0$$

$$m^* \cdot d^j = (m^* \cdot \bar{d})^2 \equiv \mu^{(s)} .$$

With $\mu^{(s)} = (K/s)^2$ for $s > 0$.
and $\mu^{(s)} = 0$ for $s = 0$. Here we have specialized to $p = 2$.

The change in the synaptic strengths as a result of these vectors entering is:

$$\dot{m} = -\alpha_\alpha (m \cdot d^i) d^i , \qquad d^i \in \mathcal{D}^\alpha$$

$$\dot{m} = +\alpha_\mu (m \cdot d^j - (m \cdot \bar{d})^2) d^j , \qquad d^j \in \mathcal{D}^\mu .$$

m is very near the fixed point, so that $m = m^* + x$. If $\mathcal{D}$ forms a complete set (i.e., K = N) then we can expand x in the set $\mathcal{D}$:

$$x = \sum_{\ell=1}^{K} x_\ell d^\ell .$$

Thus the evolution of the perturbation is given by (m = x):

$$\left.\begin{aligned} \dot{x} &= -\alpha_s (x\cdot d^i) d^i \\ \dot{x} &= -\alpha_\mu (x\cdot d) d^j + O(x^a) \end{aligned}\right\} \qquad s > 0$$

where $d = d^j(2/s - 1) + 2/s \sum_{\ell \neq j}^{K} d^\ell$.

$$\dot{x} = \alpha\ (x\cdot d^i)^2 d^i, \quad \text{for } i = 1,2,\ldots,K. \qquad s = 0$$

Now $x = \sum_{\ell=1}^{K} x_\ell d^\ell$. Thus $\dot{x} = \sum_{\ell=1}^{K} \dot{x}_\ell d^\ell$. For the K linearly independent vectors, the coefficient of each vector must vanish separately. Thus the expectation value over the environment yields:

$$\left.\begin{aligned} \dot{x}_i &= -\alpha_s (x\cdot d^i) \\ \dot{x}_j &= -\alpha_\mu (x\cdot d) \end{aligned}\right\} \qquad s > 0$$

$$\dot{x}_i = \alpha\ (x\cdot d^i)^2 \qquad s = 0$$

To illustrate we consider the simplest environment: a set of K = N orthonormal vectors, $\mathcal{D} = \{d^1, \ldots, d^K\}$; Then

$$\left.\begin{aligned} \dot{x}_i &= -\alpha_s x_i \\ \dot{x}_j &= -\alpha_\mu (2/s - 1) x_j - \alpha 2/s \sum_{q}{}' x_q \end{aligned}\right\} \qquad s > 0$$

$$\dot{x}_i = \alpha (x_i)^2 \qquad s = 0$$

where $\sum_q'$ means a sum over all q such that $d^q \in \mathcal{D}^\mu$.

We see immediately that for s = 0, the perturbation x grows faster than exponentially, and m* is an unstable fixed point. If s > 0, then $x_i = x_i(0) e^{-\alpha_\nu t}$ and those components of the perturbation along the vectors for which $m^* \cdot d = 0$ will decay exponentially. What of the components of x along the 'preferred' vectors for which $m^* \cdot d = (m^* \cdot \bar{d})^2$? Solving the second equation gives for s = 1 and $\alpha_\nu \neq \alpha_\mu$:

$$x_j(t) = (x_j(0) - \frac{2\alpha_\mu}{\alpha_\nu - \alpha_\mu} X) e^{-\alpha_\mu t} + \frac{2\alpha_\mu}{\alpha_\nu - \alpha_\mu} X e^{-\alpha_\nu t}.$$

And for the special case of $\alpha_\nu = \alpha_\mu$:

$$x_j(t) = (x_j(0) - 2\alpha_\nu X t) e^{-\alpha_\nu t}.$$

We have made the definition that $X = \sum_q' x_q(0)$. Thus for s = 1, all components of the perturbation will vanish as t tends to ∞. The s = 1 point, that point for which $c = \bar{c}^2$ for one pattern and c = 0 for all other patterns, is a stable point.

Consider now s = 2. For this value of s, two patterns give a non-zero response, and all others give a zero response. Then $\mathcal{D}^\mu = \{d^1, d^2\}$ and $\mathcal{D}^\nu = \{d^3, \ldots, d^K\}$. The solutions of the differential equations for the components of x along d^1

and d^2 are (for $\alpha_o \neq \alpha_\mu$):

$$x_1(t) = \tfrac{1}{2}(x_1(0) - x_2(0))e^{\alpha_\mu t} + \tfrac{1}{2}(x_1(0) + x_2(0) - \frac{2\alpha_\mu}{\alpha_o - \alpha_\mu}X)e^{-\alpha_\mu t} + \frac{\alpha_\mu}{\alpha_o - \alpha_\mu}e^{-\alpha_o t},$$

$$x_2(t) = \tfrac{1}{2}(x_2(0) - x_1(0))e^{\alpha_\mu t} + \tfrac{1}{2}(x_2(0) + x_1(0) - \frac{2\alpha_\mu}{\alpha_o - \alpha_\mu}X)e^{-\alpha_\mu t} + \frac{\alpha_\mu}{\alpha_o - \alpha_\mu}e^{-\alpha_o t},$$

and for $\alpha_o = \alpha_\mu$:

$$x_1(t) = \tfrac{1}{2}(x_1(0) - x_2(0))e^{\alpha_o t} + \tfrac{1}{2}(x_1(0) + x_2(0) - \alpha_o Xt)e^{-\alpha_o t},$$

$$x_2(t) = \tfrac{1}{2}(x_2(0) - x_1(0))e^{\alpha_o t} + \tfrac{1}{2}(x_2(0) + x_1(0) - \alpha_o Xt)e^{-\alpha_o t}.$$

Thus only in the special case of $x_1(0) = x_2(0)$ will the $s = 2$ point be stable. However this means that if the perturbation is only slightly off the line in m-space on which $x_1 = x_2$, then the perturbation will grow rapidly. This simply means that the $s = 2$ point is a saddle point. Further analysis for $s > 2$ yields similar results; for $s > 2$, the fixed points are unstable. Our linear analysis indicates that for the case of an orthonormal environment, only the $s = 1$ point is stable asymptotically.

This form of analysis can be carried out for the general linearly-independent environment in two dimensions. The results found above hold for this general environment: only the $s = 1$ point is stable asymptotically. The analysis for the non-orthogonal, K-dimensional environment is currently under investigation.

These same methods may be used to illustrate the correlation between ocular dominance and selectivity in the monocular deprived environment. Following the arguments presented in Bienenstock et al. (1982) we consider an MD environment specified by the circular environment d_r for the right eye and n a "pure noise vector for the left eye. We now assume the cell has evolved to the state given by $\phi(m_r^*, 0)$ where m_r^* is a stable selective state in the environment d_r. As before, we consider a perturbation from the fixed point; the state of the cell now is given by $\phi(m_r^* + x_r, x_\ell)$. The perturbation then evolves according to (where the noise is assumed to have zero mean):

$$\dot{x}_r = (m_r^* \cdot d_r + x_r \cdot d_r + x_\ell \cdot n, m^* \cdot \bar{d}_r + x_r \cdot \bar{d}_r) d_r$$

$$\dot{x}_\ell = (m_r^* \cdot d_r + x_r \cdot d_r + x_\ell \cdot n, m_r^* \cdot \bar{d}_r + x_r \cdot \bar{d}_r) n.$$

The stability of the equation for x_r has already been assumed in the conditions a on m_r^*. The equation for x_ℓ is analyzed by dividing the right eye inputs into those for which $\phi(m_r \cdot d_r, m_r \cdot \bar{d}_r)$ is far from zero (either above or below) and those for which $\phi(m_r \cdot d_r, m_r \cdot \bar{d}_r) = 0$. For those vectors in the first class, the sign of ϕ is determined by d_r alone, hence the equation for x_ℓ is the equation of a random walk Those vectors in the second class elicit a response near the threshold θ , or near zero. Using the methods described above, it is easy to see that vectors with a response near θ cause the perturbation to evolve according to:

$$\dot{x}_\ell \sim \alpha_\mu (x_\ell \cdot n) n$$

and those for which $m_\mu^* \cdot d_\nu \simeq 0$ cause the perturbation to evolve like:

$$\dot{x}_i \sim -\alpha_0 (x_i \cdot n) n$$

where α_0 and α_μ are defined above. Finally, averaging these equations (the distribution of n is assumed symmetric with respect to x_i) yields, respectively

$$\dot{x}_i \sim \alpha_\mu \overline{n_0^2}\, x_i$$

$$\dot{x}_i \sim -\alpha_0 \overline{n_0^2}\, x_i$$

where $\overline{n_0^2}$ is the average squared magnitude of the noise input to a single synapse from the closed eye.

Now for the case of K linearly independant vectors defining the environment d_ν, only vectors of the second class enter the system after $(m_\nu^*, 0)$ has been reached. Also, for m_ν^* a stable selective state (i.e., s = 1), only one vector of d_ν will elicit a response near threshold. Thus the results above show that only one input will cause the perturbation to grow and all others tend to return the state of the cell to $(m_\nu^*, 0)$. For the general environment, the more selective m_ν^* is with respect to d_ν, the greater the number of inputs that will drive $(m_\nu^* + x_\nu, x_i)$ to $(m_\nu^*, 0)$. This linear analysis then allows us to conclude that under proper conditions on ϕ and (d_ν, n), (Bienenstock et al., 1982), the state $(m_\nu^*, 0)$ is stable on the average and the fluctuation of x_i due to 'preferred' patterns is smaller for more selective m_ν^*.

REFERENCES

Elie L. Bienenstock, "A Theory of Development of Neuronal Selectivity", Ph.D. Thesis, Division of Applied Mathematics and Center for Neural Science, L. N Cooper, Thesis Supervisor, June 1980.

Bienenstock, E., "A theory of development of neuronal selectivity," Doctoral thesis, Brown University, Providence, Rhode Island (1980).

Bienenstock, E. L., L. N Cooper, and P.W. Munro, "Theory for the development of neuron selectivity: Orientation selectivity and binocular interaction in visual cortex", Jour. of Neurosci. 2:32-48, (1982).

Blakemore, C. and D. E. Mitchell, "Environmental modification of the visual cortex and the neural basis of learning and memory," Nature 241:467 (1973).

Blakemore, C. and R. C. Van Sluyters, "Reversal of the physiological effects of monocular deprivation in kittens. Further evidence for a sensitive period." J. Physiol. (London) 237:195-216 (1974).

Blakemore, C. and R. C. Van Sluyters, "Innate and environmental factors in the development of the kitten's visual cortex," J. Physiol. (London) 248:663-716 (1975).

Buisseret, P. and M. Imbert, "Visual cortical cells. Their developmental properties in normal and dark reared kittens," J. Physiol. (London) 255:511-525 (1976).

Buisseret, P., E. Gary-Bobo, and M. Imbert, "Ocular motility and recovery of orientational properties of visual cortex neurons in dark reared kittens," Nature 272:816-817 (1978).

Cooper, L. N, F. Liberman and E. Oja, "A theory for the acquisition and loss of neuron specificity in visual cortex, Biol. Cybernetics 33:9-28 (1979).

Creutzfeldt, O. D., U. Kuhnt, and L. A. Benevento, "An intracellular analysis of visual cortical neurones to moving stimuli: Responses in a cooperative neuronal network," Exp. Brain Res. 21:251 (1974).

Davis, T. L. and P. Sterling, "Microcircuitry of cat visual cortex: Classification of neurons in layer IV of area 17, and identification of the patterns of lateral geniculate input," J. Comp. Neurol. 188:599 (1979).

Fregnac, Y. and M. Imbert, "Cinetique de developpement du cortex visuel," J. Physiol. (Paris) 6:Vol. 73 (1977).

Fregnac, Y., "Cinetique de developpement du cortex visuel primaire chez le chat. Effets de la privation visuelle binoculaire et modele de maturation de la selective a

l'orientation," Doctoral thesis, University Rene Descartes (1978).

Fregnac, Y. and M. Imbert, "Early development of visual cortical cells in normal and dark-reared kittens: Relationship between orientation selectivity and ocular dominance," J. Physiol. (London) 278:27-44 (1978).

Hebb, D. O., The Organization of Behavior. Wiley, New York, p.62 (1949).

Hornung, J. P. and L. J. Garey, "The Thalamic Projection to Cat Visual Cortex: Ultrastructure of Neurons Identified by Golgi Impregnation or Retrograde Horseradish Peroxidase Transport," Neuroscience 6:1053 (1981).

Hubel, D. H. and T. N. Wiesel, "Receptive fields of single neurons in the cat striate cortex," J. Physiol. (London) 148:574-591 (1959).

Imbert, M. and Y. Buisseret, "Receptive field characteristics and plastic properties of visual cortical cells in kittens reared with or without visual experience," Exp. Brain Res. 22:2-36 (1975).

Kasamatsu, T. and J. D. Pettigrew, "Depletion of brain catecholamines: failure of ocular dominance shift after monocular occlusion in kittens," Science 194:206-209 (1976).

Kasamatsu, T. and J. D. Pettigrew, "Preservation of binocularity after monocular deprivation in the striate cortex of kittens treated with 6-hydroxydopamine," J. Comp. Neurol. 185:139-162 (1979).

Kohonen, T., Associative Memory: A System Theoretic Approach, Springer-Verlag, Berlin (1977).

Kratz, K. E. and P. D. Spear, "Effects of visual deprivation and alterations in binocular competition on responses of striate cortex neurons in the cat," J. Comp. Neurol. 170:141 (1976).

Leventhal, A. G. and H. V. B. Hirsch, "Receptive field properties of different classes of neurons in visual cortex of normal and dark-reared cats," J. Neurophysiol. 43:1111 (1980).

Nass, M. M. and L. N Cooper, "A theory for the development of feature detecting cells in visual cortex," Biol. Cybernetics 19:1-18 (1975).

Perez, R., L. Glass, and R. J. Shlaer, "Development of specificity in the cat visual cortex," J. Math. Biol. 1:275 (1975).

Sillito, A. M., "The contribution of inhibitory mechanisms to the receptive field properties of neurons in the cat's striate cortex," J. Physiol. 250: 304-330 (1975).

Von der Malsburg, C., "Self-organization of orientation sensitive cells in the striate cortex," Kybernetik 14:85 (1973).

Watkins, D. W., J. R. Wilson, and S. M. Sherman, "Receptive field properties of neurons in binocular and monocular segments of striate cortex in cats raised with binocular lid suture," J. Neurophysiol. 41:322 (1978).

Wiesel, T. N. and D. H. Hubel, "Single-cell responses in striate cortex of kittens deprived of vision in one eye," J. Neurophysiol. 26:1003-1017 (1963).

Wiesel, T. N. and D. H. Hubel, "Comparisons of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens," J. Neurophysiol. 28:1029-1040 (1965).

Figure Captions

Figure 1. Schematic representation of an LxN array of ideal synaptic junctions. The value M_{ij} gives the net influence of the j-th afferent on the i-th cell.

Figure 2. Examples of aspecific (A), immature (I), and specific (S) response curves.

Figure 3. Distribution of the different types of cells in three age groups in the normally reared kittens (upper part) and in the dark-reared kittens (lower part). The ordinate is normalized so that the heights are the percentages of cells in the various function groups. Type 1, nonactivatable (); 2, nonspecific (); 3, immature(); 4, specific (). (From Fregnac and Imbert, 1977, 1978.)

Figure 4. Evolution of the development of the various specificity groups in cats raised (a) normally and (b) in total darkness. The results are based on an unweighted regression analysis of 1050 cells (From Fregnac, 1978.)

Figure 5. Distribution, in percentage, of the three types of visual cortical units (area 17) recorded after 6 hours of visual exposure for

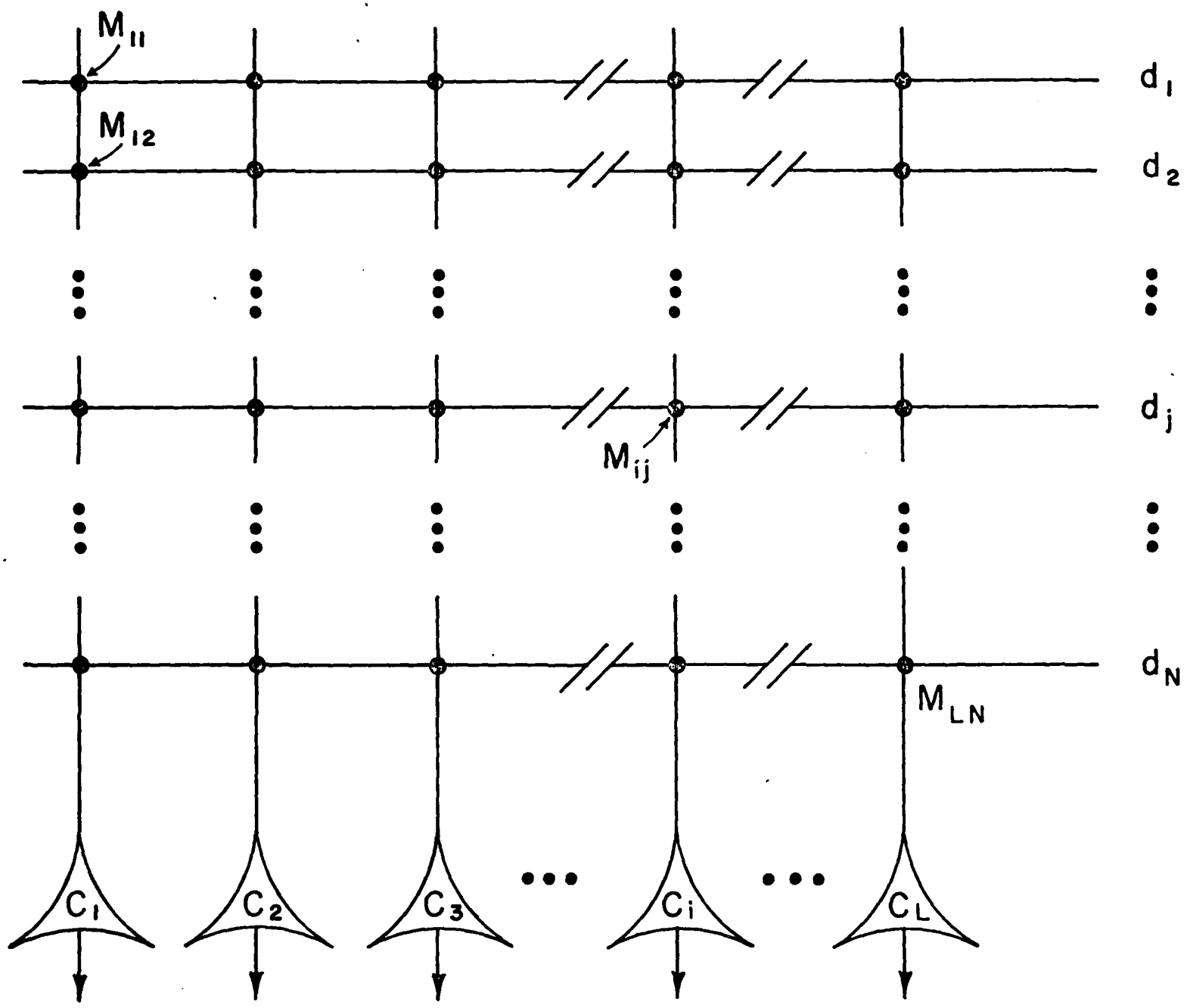
6-week-old dark reared kittens. Columns: I, dark-reared kittens; IV, normally-reared kittens. During 6 hours of exposure, conditions were: in II and III, freely moving; in III, 12 hours in the dark followed the 6 hours of exposure. Numbers of visual cells recorded are given under each column. Specific cells (▩) are activated by oriented stimuli within a sharp angle (<60°). Immature cells (▨) are activated by oriented stimuli within a larger angle (<150°). Nonspecific cells (□) are activated by nonoriented stimuli moving in any direction. A statistical analysis reveals no significant difference in the percentages of immature and specific units in between columns III and IV. Therefore it may be that for a 6-week-old dark-reared kitten, a 6-hour exposure to visual input followed by 12 hours in the dark is sufficient to produce a distribution of cortical cells similar to that of normally reared animals. (From Buisseret, Gary-Bobo, and Imbert, 1978.)

Figure 6. The modulatory function $\phi(c,\theta(\vec{c}))$ is shown for two values of $\theta(c)$. As $\vec{c}$ increases, so does θ. The modification is (anti-) hebbian ((anti-) parallel to the input vector d) if the evoked response c is greater (less) than θ.

Figure 7. The state space of a two-synapse cell in a two-pattern environment is shown. For clarity, the vectors are chosen to lie along the axes ($d^1=(1,0)$ and $d^2=(0,1)$), but this is not necessary (see figure 3 in Bienenstock et al., 1982). (a). The directions of change in the state m as a result of each input (open arrowheads for

d^1, solid for d^2) are shown for each region and on the isoclines which separate them. Each fixed point is labelled by its order s. One can see that along the isoclines, $\dot{\phi}=0$ with respect to one of the patterns. The s=2 point is a saddle point: it attracts trajectories from some directions, but repels them in others, so it is not stable. The s=0 point repels all nearby states. Hence only the s=1 points are stable. (b). Some sample trajectories are shown with the isoclines (bold).

Figure 8. Results of machine simulation of neuron development in four experimental paradigms: (a) Normal Rearing, (b) Binocular Deprivation, (c) Monocular Deprivation, and (d) Reverse Suture. Each figure shows the response vs. orientation of the test stimuli to each eye both before (dashed) and after (solid) a period of plastic development.



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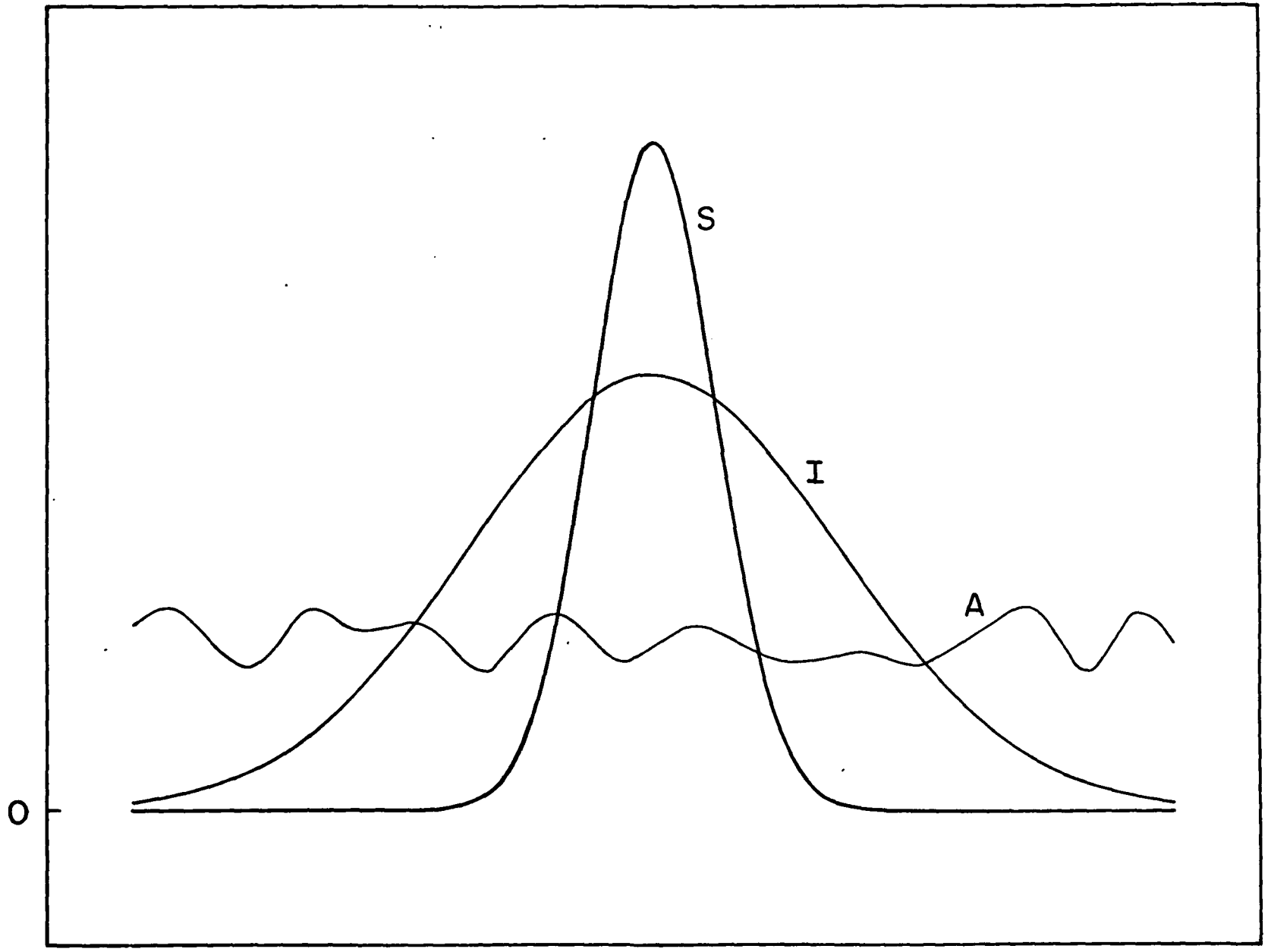


Fig. 2

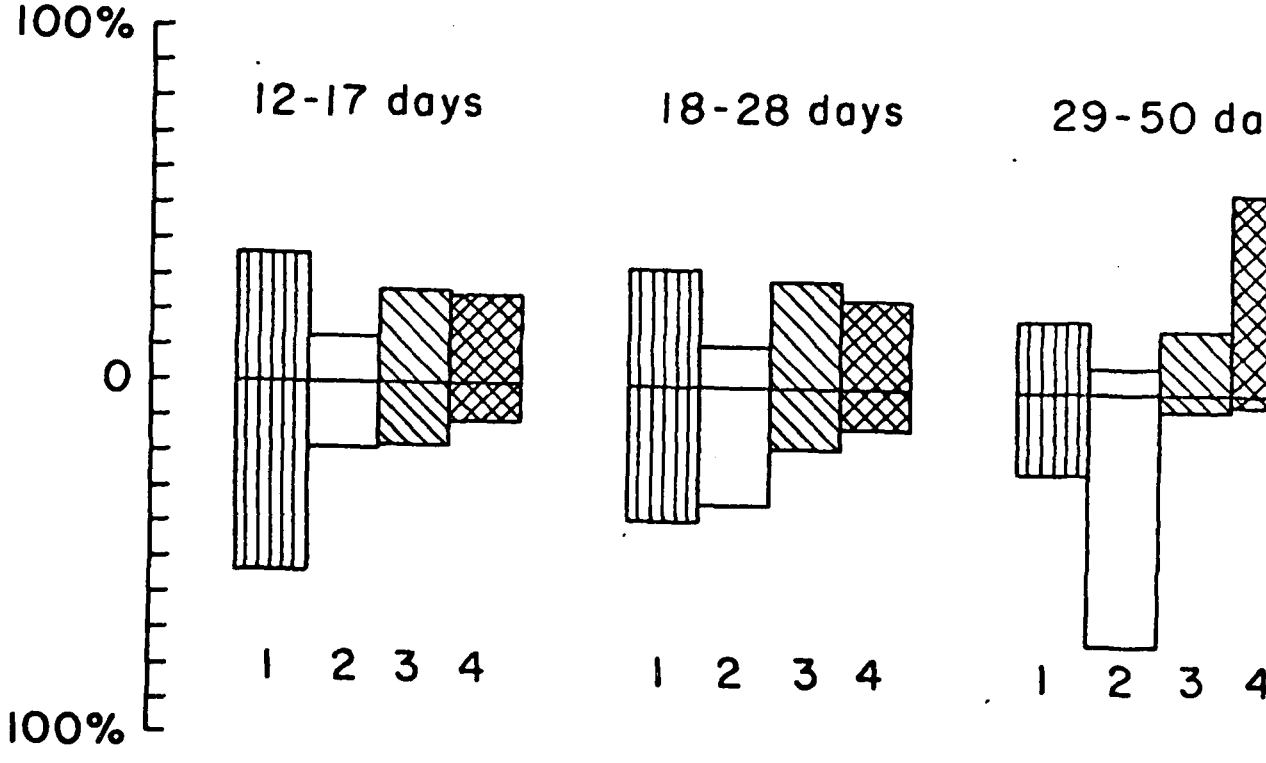

100%
0
100%
12-17 days
18-28 days
29-50 days
1 2 3 4
1 2 3 4
1 2 3 4
Normally reared kittens
Dark reared kittens

Fig. 3

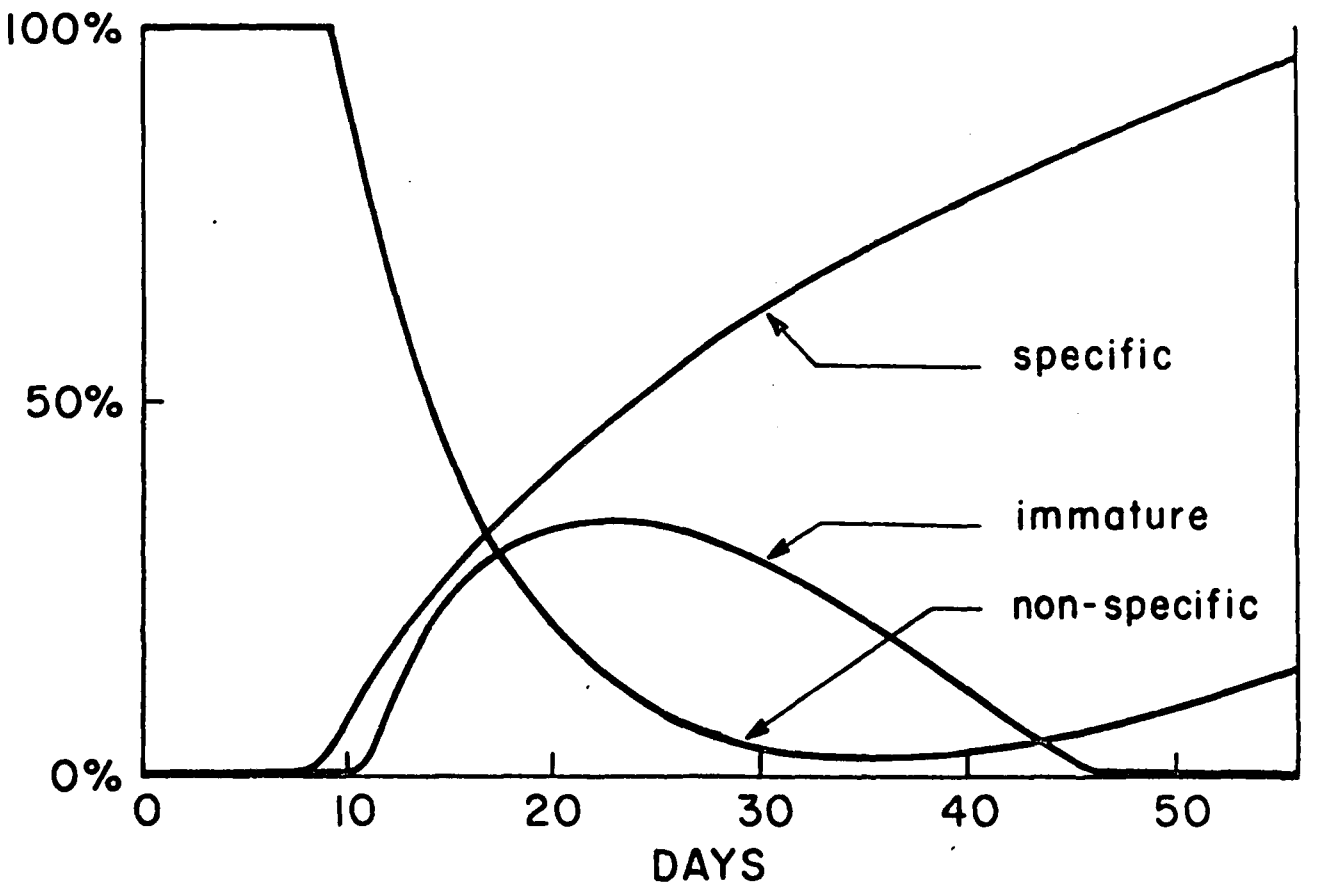


Fig. 4a

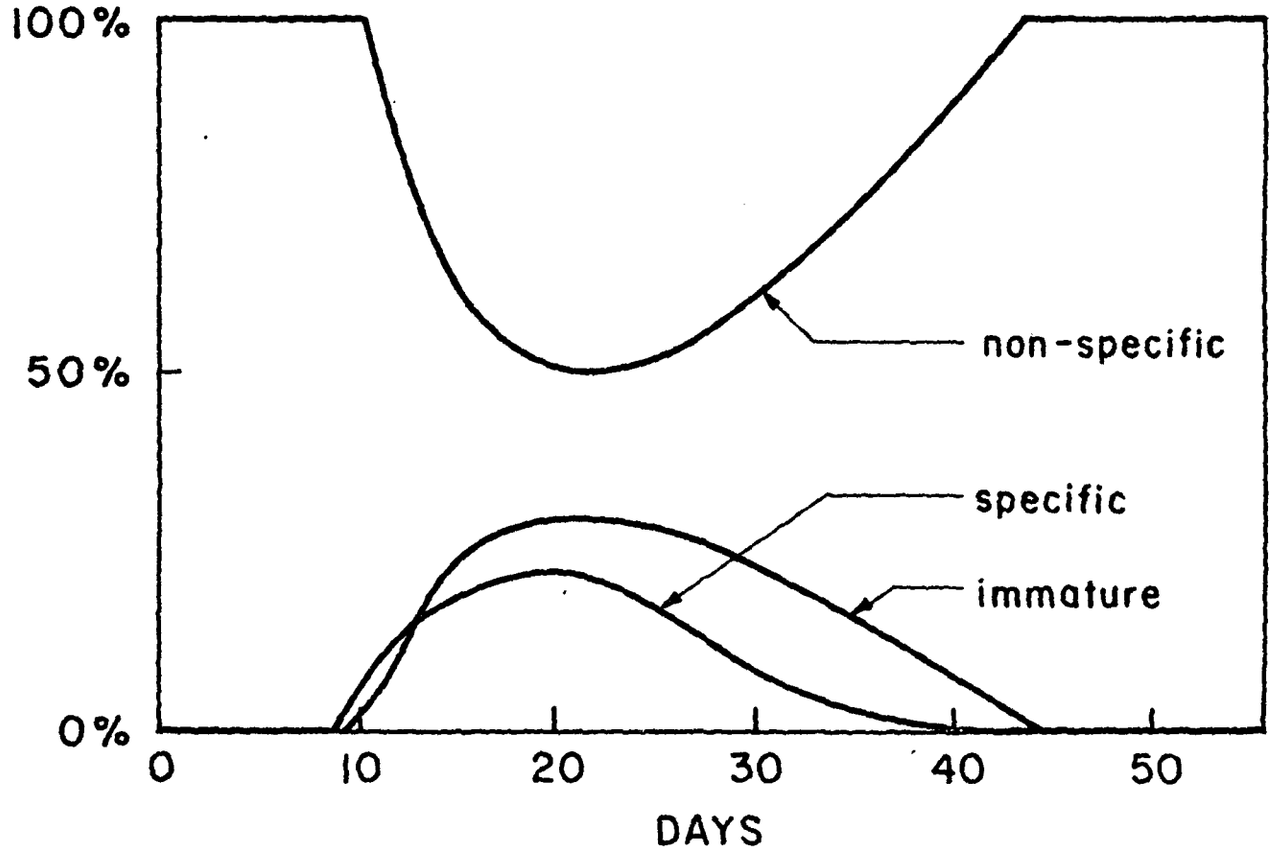


Fig. 4b

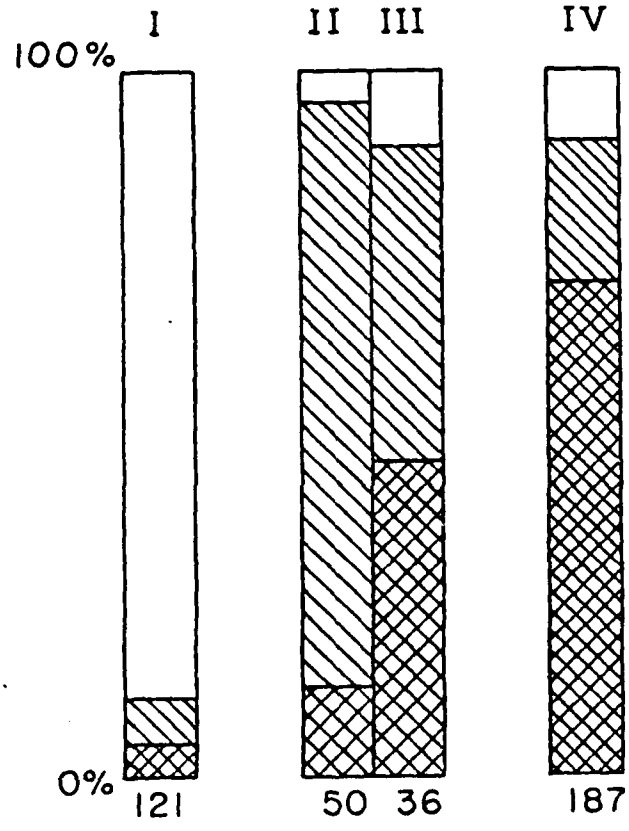
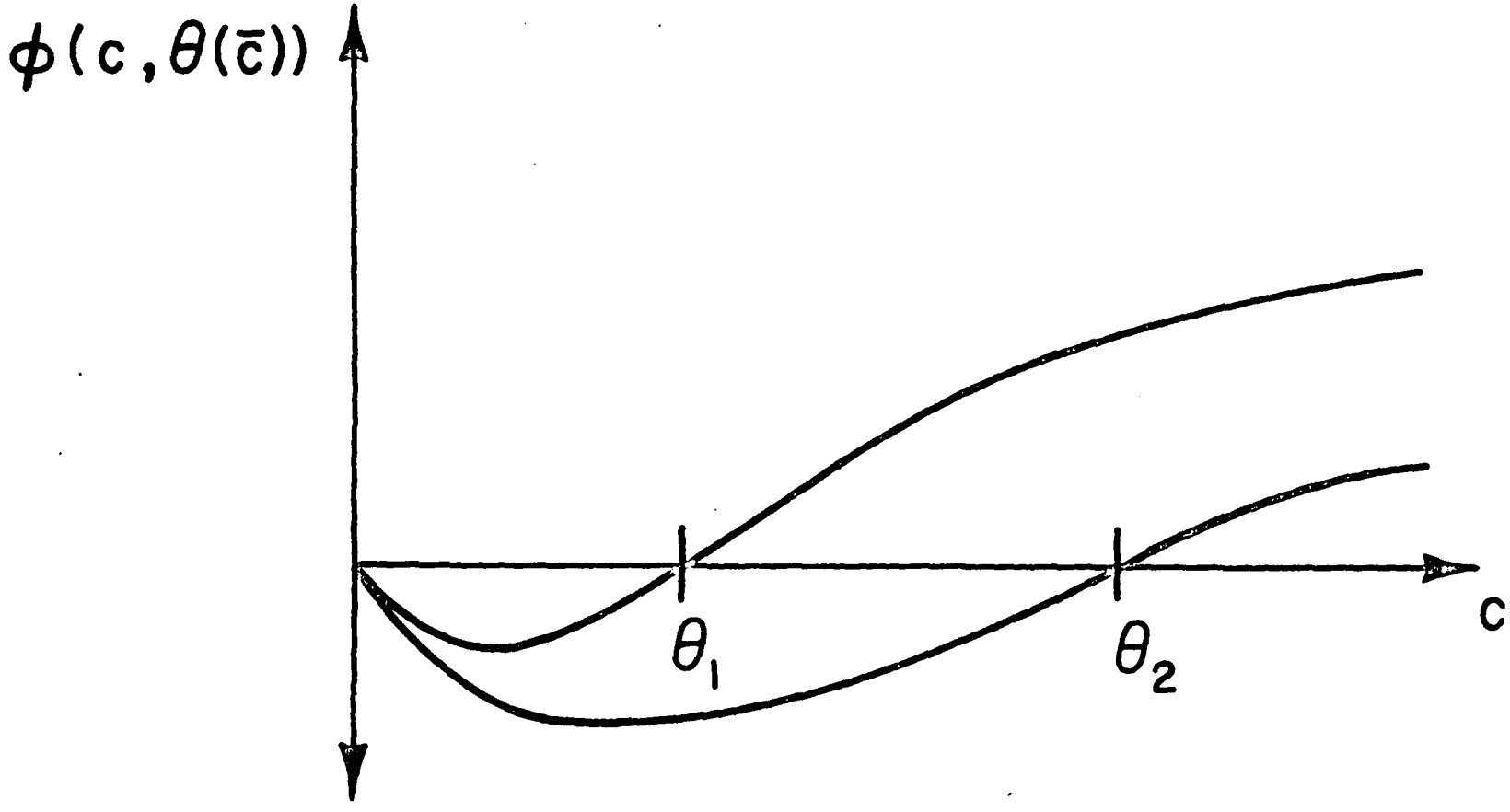


Fig. 5



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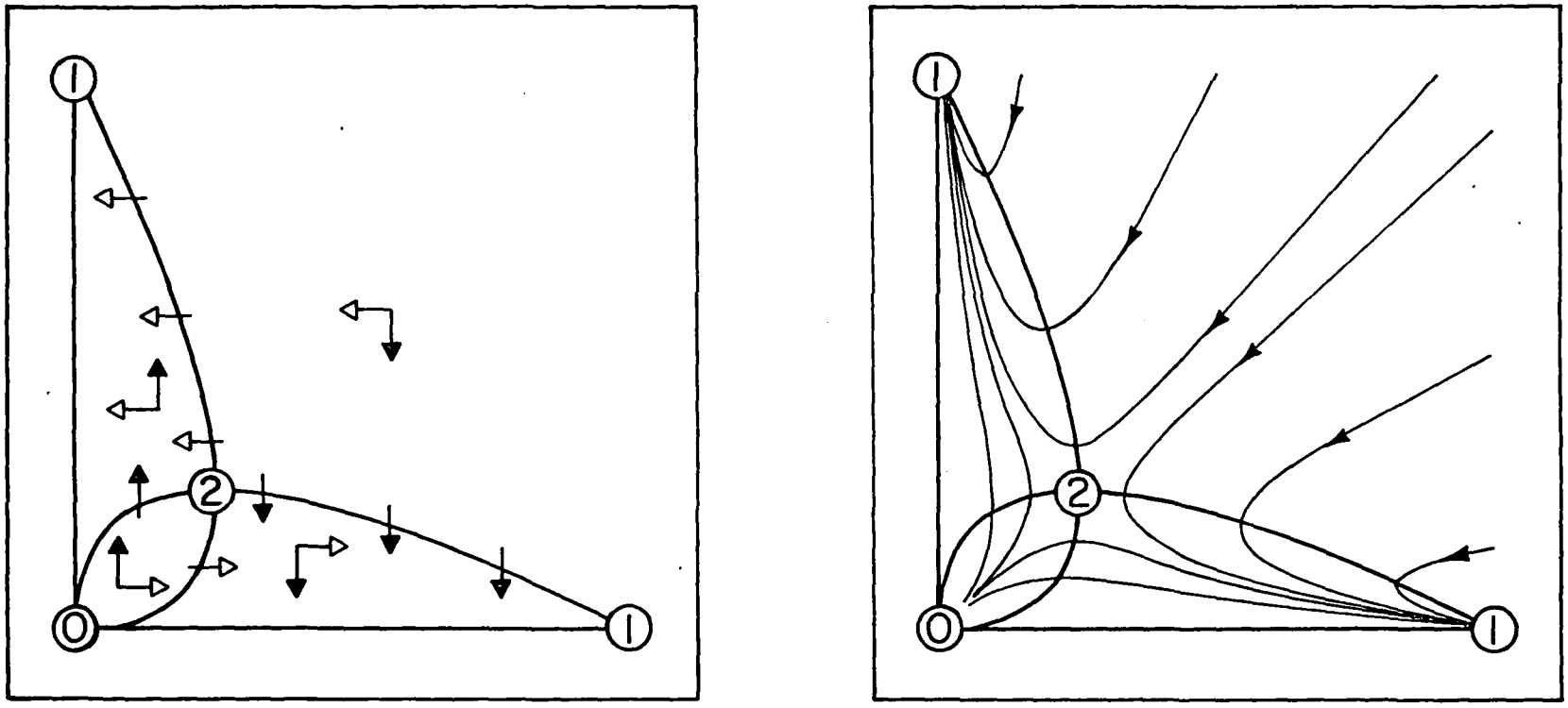
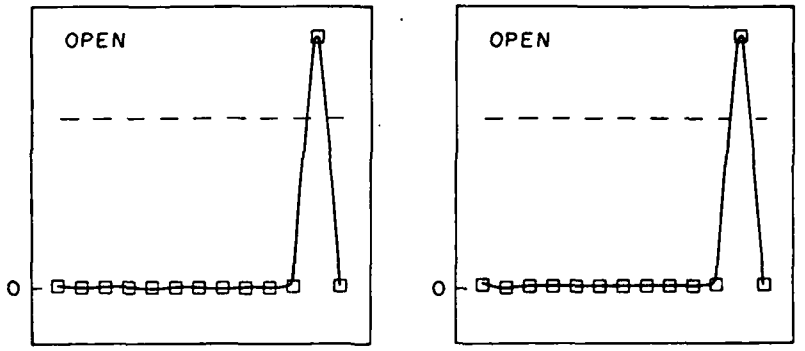


(a) (b)

Fig. 7



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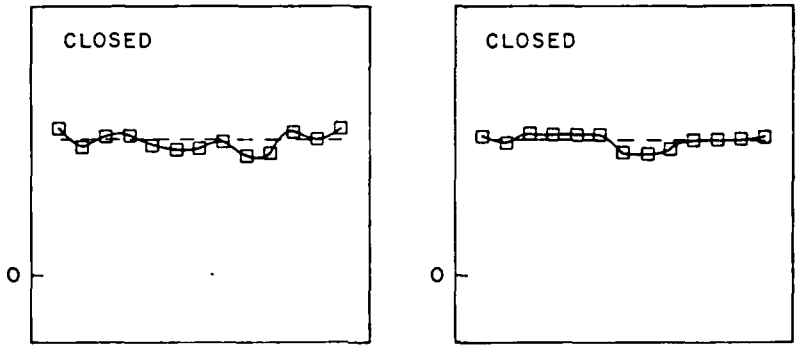

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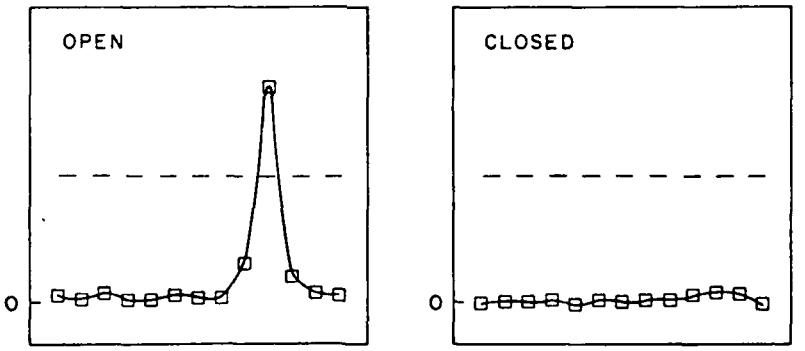
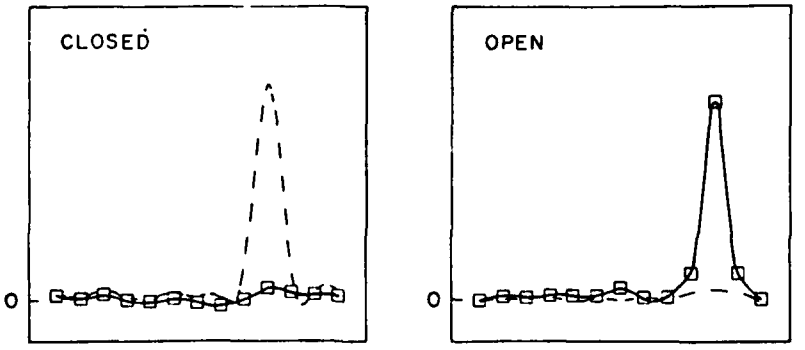
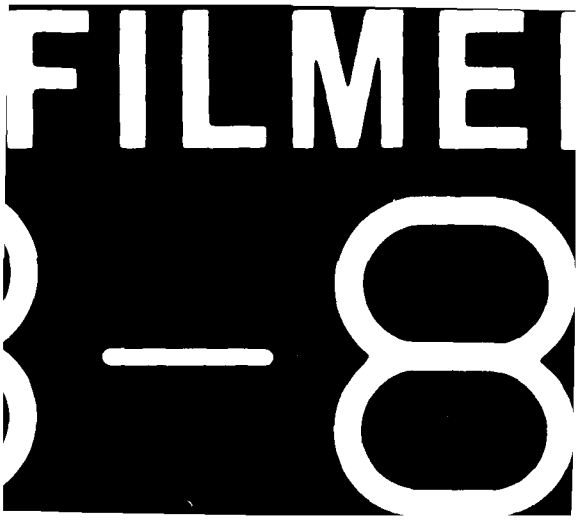


(c) MD



(d) RS

Fig. 8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